Modern Concepts of Cardiovascular Disease

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NOMENCLATURE IN CORONARY ARTERY DISEASE

At the present time there is considerable confusion concerning terminology in coronary artery disease. A number of expressions exist, including angina pectoris, coronary occlusion and coronary thrombosis, coronary insufficiency, coronary failure and myocardial infarction. These terms are often used loosely and are given various connotations. Recently it has been suggested that the terms coronary occlusion and thrombosis be superseded by coronary insufficiency and coronary failure. We believe that "coronary failure," in addition to being new, has the disadvantage of being ambiguous. The term coronary occlusion should be retained because it represents a definite clinical and electrocardiographic entity and is firmly established in usage. The same is true of angina pectoris due to coronary disease. Coronary insufficiency is a useful term when restricted to necrosis or infarction of the heart muscle without complete occlusion. To be sure, in a general way, coronary insufficiency may be considered to include every interference with the coronary circulation, whether it is transient and on a functional basis, as in a simple attack of angina pectoris, or whether it is caused by complete obstruction of a coronary artery, i.e., by coronary occlusion. It would be preferable, in our opinion, to continue the use of the terms angina pectoris and coronary occlusion and to limit coronary insufficiency in the sense already mentioned. We shall now define these three terms in detail and emphasize their differential diagnosis.

Angina pectoris due to coronary disease is a transitory attack of substernal pain precipitated by exertion, emotion, ingestion of food, cold, tobacco, adrenalin, insulin. It is due to a temporary ischemia and is not accompanied by any acute pathological changes in the myocardium. The pain usually lasts a few seconds to a few minutes, rarely one or two hours. Frequently relief is afforded merely by ceasing to walk. The pain varies from an agonizing constriction to a vague pressure and frequently is radiated particularly into the arms or neck; it may be felt as a choking sensation. The pain is relieved by nitroglycerin. Signs of shock are usually absent except in severe attacks. Vomiting does not occur. Changes in the heart sounds, heart failure, arrhythmias and a drop in blood pressure are absent; the blood pressure remains unchanged or may rise. The electrocardiagram is unaltered or shows transient depression of the RS-T interval and inversion of the T-wave in one or more leads. Elevation of the RS-T transition is practically never observed. When changes in the electrocardiogram occur they are usually very transient. If they persist for any length of time we are probably dealing with more than merely an attack of angina pectoris, namely, ischemia severe enough to produce anatomic alterations in the myocardium. This is described later as acute coronary insufficiency.

Coronary occlusion or thrombosis, with myocardial infarction, is another characteristic syndrome and presents specific electrocardiographic changes. Occlusion is a better term than thrombosis since it has been clearly demonstrated that the commonest mechanism of complete occlusion of the lumen of a coronary vessel is intimal hemorrhage into a sclerotic plaque with hematoma into the vessel wall and secondary thrombosis on the injured intima. At postmortem one usually finds multiple recent or old thrombi although the former are not of exactly the same age. Coronary sclerosis and hypertension predispose to coronary occlusion but the acute attack occurs irrespective of external factors, such as effort and excitement. The majority of attacks occur at rest or during sleep. The pain of coronary occlusion is usually much more prolonged than that of angina pectoris and may persist for hours in spite of the administration of morphine. It is often referred to the epigastrium. It is unaffected or even aggravated by nitroglycerin, which may be dangerous since it lowers the blood pressure and increases the work of the heart; as a result it may increase the tendency to shock. Frequently premonitory symptoms presage the occurrence of coronary occlusion. The interval may be a few hours, days or even weeks. The sudden onset of a persistent anginal syndrome or an increase in the severity of the pain may indicate the advent of an occlusion. Shock, which is rare in an anginal attack, is a classical accompaniment of coronary occlusion; it usually occurs at the onset but may be delayed. Unlike angina pectoris, gastrointestinal symptoms, such as nausea and vomiting, are very common in coronary occlusion and may be aggravated by morphine. These symptoms, when associated with epigastric pain and tenderness, may simulate an "acute abdomen." The blood pressure invariably falls in coronary occlusion although it may be temporarily increased during the period of pain and in hypertensives may not fall for several days. A change in heart sounds is a cardinal sign of acute coronary occlusion; the commonest is a reduction in intensity of the first sound at the apex, due to a diminution in amplitude, i.e., intensity, of the high frequency component. As a result the second sound is louder than the first. Presystolic gallop rhythm, due to an accentuated auricular sound, is heard in fully half the cases associated with the left heart failure. A pericardial rub is present in 10 to 20% of cases. Left heart failure is very common, right heart failure less so. The patient with coronary occlusion requires bed rest for one to two months but the majority survive the first attack. Rehabilitation is then gradual. A number of positive laboratory findings, such as leukocytosis, fever and later a rapid sedimentation rate confirm the diagnosis of coronary occlusion. Azotemia and glycosuria may appear or be greatly exaggerated.

The electrocardiogram shows typical, progressive changes consisting of RS-T elevations and deep Q-waves; the former progress into T-wave inversions. There is usually a reciprocal relationship of the RS-T segments and T-waves in leads I and III. In

occlusion with infarction of the anterior surface of the left ventricle RS-T elevation, T-wave inversion and deep Q-wave are present in leads I and IV whereas when the infarction involves the posterior surface these changes appear in leads II and III. Both patterns occur with equal frequency since posterior infarction occurs as often as anterior. The RS-T changes are presumably related to the myocardial infarction resulting from the occlusion, yet we have observed them within 20 minutes after the onset, before infarction has set in, and at post-mortem where a coronary occlusion existed without infarction as yet. The electrocardiogram shows a typical, progressive pattern of change. The alterations in the RS-T segment give way to fully inverted T-waves by the end of the second or third week. The electrocardiogram may begin to return toward normal beginning the fourth week but becomes complete only in approximately 10% of patients, usually between the third month and the end of the first year. Q-waves are the most persistent abnormality. We wish to emphasize the specificity of this electrocardiogram. Although its presence indicates an acute coronary occlusion it is not to be inferred that the converse is true, namely, that every case of coronary occlusion produces this typical picture. For example, the presence of a hypertensive electrocardiogram, i.e., S-T depression and T-wave inversion in lead I, slight S-T elevation in lead III and even a deep Q_3 may prevent the appearance of typical changes. This is true also of bundle-branch or interventricular block. Furthermore, if the patient has had previous coronary occlusions, changes due to these may confuse the picture. The occurrence of certain arrhythmias, for example, ventricular tachycardia, while often diagnostic of acute coronary occlusion, also may alter the appearance of the electrocardiogram.

The specific electrocardiographic pattern described above is associated with a confluent, massive infarct extending from the endocardium to the pericardium and thus frequently giving rise to embolization and pericarditis.

Coronary occlusion is thus a specific anatomic entity occurring in the progressive course of coronary sclerosis, irrespective of external factors. It is associated with a characteristic clinical syndrome and a specific electrocardiogram, consisting of deep Q-waves and RS-T elevations progressing into T-wave inversions.

In between these two groups falls acute *coronary insufficiency*. As a rule it is associated with a precipitating factor which decreases the coronary flow, such as tachycardia, heart failure, heart block, acute hemorrhage, shock, operation, acute obdominal conditions, trauma, pulmonary embolism and cor pulmonale, or which increases the work of the heart and the oxygen requirement of the heart muscle, such as hypertensive crises, aortic stenosis, post-operative complications and infections. Predisposing factors are coronary sclerosis, hypertension, cardiac enlargement, aortic stenosis or insufficiency and heart failure. Occasionally no obvious cause is present.

Although a good deal is being written in this country on coronary insufficiency at the present time and a tremendous number of references has been present in the German literature for years, certain symptoms, signs and laboratory findings have not been clearly established. The clinical picture may closely resemble that of coronary occlusion or pain and signs of shock may be absent or inconspicuous. In fact, the diagnosis may be purely an electrocardiographic one. Heart failure, loss of intensity of the first heart sound, gallop rhythm, arrhythmias and fall in blood pressure are less common than in coronary occlusion. The lungs may be congested. Leukocytosis, fever and increased sedimentation rate are usually present to some degree. Whether azotemia or glycosuria occurs has not been definitely shown. The duration of the bout of coronary insufficiency may be brief or vary from a few hours to a few days. The course and prognosis depend in great part on the exciting cause. Death, of course, may take place.

Electrocardiographically, coronary insufficiency is characterized by depression of the RS-T segment of 1 mm. or more and T-wave inversion in two or more leads. The changes occur with equal frequency in leads 1, 2 and 3. Q-waves and RS-T elevation are very exceptional. The abnormalities are usually maximal in the initial record and progress rarely. They return to normal when the factor precipitating the coronary insufficiency has disappeared. Therefore, even when acute insufficiency simulates coronary occlusion with infarction clinically, the two conditions may be distinguished by the electrocardiographic changes. Coronary insufficiency also differs from coronary artery occlusion pathologically. If the factor producing coronary insufficiency is slight or is exerted briefly there may be no changes in the myocardium. If the ischemia is severe and prolonged it may result in focal, disseminated necrosis in the subendocardium and papillary muscles. There is good evidence that the presence of elevation of the RS-T segment in coronary occlusion is due to pericarditis or to involvement of the outer portion of the heart muscle, or both. In involvement of the subendocardial layer of heart muscle, on the other hand, RS-T depression is produced. Thus the localization of the pathological process in the subendocardial layer in coronary insufficiency explains the presence of RS-T depression in the electrocardiogram and the absence of pericarditis.

In summary, acute coronary insufficiency is a syndrome of more prolonged, or more severe myocardial ischemia than occurs in angina pectoris. It may be precipitated by many factors. The clinical picture may simulate coronary occlusion or there may be no symptoms at all but the electrocardiogram and the pathological changes are specific. The electrocardiogram is characterized by RS-T depressions and T-wave inversion in one or more leads.

If the distinctions made above are observed, there need be no confusion in the terminology of acute coronary artery syndromes. Coronary occlusion represents a classic picture; the diagnosis is apparent clinically in the presence of the symptoms and signs described and becomes certain when the specific electrocardiographic changes occur. The diagnosis of coronary insufficiency should be made when a precipitating factor exists and RS-T depressions and T-wave changes only are present, even when the clinical picture suggests coronary occlusion. Naturally these rules do not hold in every case. In persons who have suffered previous coronary closures and have an abnormal electrocardiogram, a fresh attack of occlusion may not be accompanied by typical recent changes in the electrocardiogram. Even more rarely, the electrocardiogram may show some RS-T elevation or a Q-wave when post-mortem examination reveals coronary insufficiency. However, these cases are exceptional. We believe that when the diagnosis of coronary occlusion is made by the typical electrocardiogram just described it will be confirmed pathologically in 95 per cent of the cases.

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